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ity in the application of generic names. The changes incident to the execution of such a reform are few and harmless in comparison with those perpetually necessary where names depend on residues. An excellent example of the workings of the method of elimination is appropriately revealed in the mazes of the recent *Mephitis-Chincha-Spilogale* discussion. Several prominent zoologists propose to settle this by a new rule* which eliminates elimination and yields definite types for a very small percentage of genera, but which constitutes a significant admission of the essential instability of residues. The new rule leads in the right direction, though it is but a short step on a long journey.

Obviously, the method of elimination lacks the definite and 'necessarily arbitrary' features without which, as Dr. Dall well says, 'stability is hopeless.' It may be true that the zoological laws 'are intended to * * * bring about stability,' but it is plain that the intention has not been rendered effective by adequate formulation. Systematists who appreciate stability may differ on details of the legislation needed to inaugurate the method of types, but they should not use stability as an argument for residues.

O. F. COOK.

WASHINGTON, Aug. 7, 1902.

SHORTER ARTICLES.

NATURE OF THE SPECIFIC BACTERIAL TOXINS.†

AFTER it had been demonstrated beyond any controversy that certain bacteria cause certain diseases, quite naturally the question was asked, How do bacteria cause disease? Several answers to this question have been offered. If the liver, spleen or kidney of a guinea-pig which has died from experimental anthrax be sectioned and examined under the microscope, the blood vessels of these organs will be found to be filled with bacilli. In many places the germs have grown so abundantly that they distend the smaller vessels. It was suggested that the anthrax bacillus causes

* SCIENCE, N. S., XVI., 114, July 18, 1902.

† Abstract of a paper presented by Dr. V. C. Vaughan before the Research Club of the University of Michigan.

disease and death by mechanically interrupting the functions of certain organs. This is known as the mechanical interference theory. It has no support in any other disease than anthrax, and consequently it cannot be accepted as a satisfactory answer to the question, How do germs cause disease? Another theory offered supposes that the bacteria cause disease by consuming the proteids of the body and thus depriving it of its sustenance. It is known that the proteids are necessary for the building up of cells, and it is also known that microorganisms feed upon proteids. This theory is untenable in the first place because many of the infectious diseases destroy life so quickly that the fatal effects cannot be presumed to be due to the consumption of any very large amount of proteid substance. Secondly, the distribution of the germs in the body is such in many diseases that they do not come in contact with any large percentage of the proteids of the body, and thirdly, the symptoms of the majority of the infectious diseases are not those which would be produced by withdrawing from the various organs their food supply. A third theory supposed that the germs cause the symptoms of the disease and death by depriving the red blood corpuscles of their oxygen. This theory was suggested by the resemblance between the symptoms of anthrax and those of carbon dioxide poisoning. More extended observation soon demonstrated the fallacy of this theory, especially inasmuch as it was shown that the amount of physiological oxidation going on in the bodies of animals sick with anthrax was not diminished by the disease. In this way the theory that germs destroy life by depriving the blood of its oxygen has been shown to be not applicable to anthrax, and in fact not to any known infectious disease. Next it was suggested that bacteria cause disease by forming chemical poisons. This is the theory which has found general acceptance and which is now generally believed to be the true explanation, although none of the specific toxins has been isolated in a state of chemical purity. The elaboration of chemical poisons by bacteria may occur in either of two ways: In the first place the bacterium, either

acting as a ferment, or by forming some soluble enzyme, may split up the proteids or other constituents of the body or of the artificial culture medium, and among these split products poisons may be produced. In this way the formation of the specific toxins would be an analytical process. In confirmation of this theory, highly poisonous bodies have been found in artificial culture media of some of the pathogenic bacteria. Some of the first of these poisonous bodies found were basic in character and were known as ptomains, which is a designation given to putrefactive alkaloids. Many chemists have sought diligently among the basic products of putrefaction for the specific toxins of the infectious diseases, and while a few highly poisonous ptomains have been found, it is safe to say that no one has yet been discovered to which all the symptoms and lesions of a disease could be attributed. The researches of Roux and Yersin, followed by those of Brieger and Fraenkel, on the diphtheria toxin, led to the conclusion that the specific poisons, instead of being basic in character, are modified proteids, and to these the term toxalbumins has been given. However, competent workers have failed to find anything like toxalbumins in cultures of many of the most virulent pathogenic bacteria. Anthrax is a disease to which all of these theories concerning the *modus operandi* of bacteria have been referred, and although Brieger and Fraenkel thought at one time that they had found a specific toxalbumin in the bodies of animals dead from anthrax, more careful investigations by other chemists have failed to confirm their results, and it is safe to say to-day that no specific bacterial toxin has been found either in the body after death from an infectious disease, or in artificial culture media; at least, no substance belonging to this group of bodies to which the symptoms and lesions of the disease could be attributed. The other possible explanation of the production of chemical poisons by pathogenic bacteria is that these substances are formed by synthetical processes and are built up in the cells of the microorganisms. The problem which Dr. Vaughan and his students have attempted to solve was that of determining

whether or not the cells of pathogenic bacteria contain specific toxins. By means of the large incubating tanks, devised by Dr. Vaughan, and which have been described in SCIENCE on page 378 of the issue of March 7, 1902, the cellular substance of the pathogenic bacteria has been obtained in large amount. The first experiments were made with the colon bacillus. A virulent form of this germ was first grown in ordinary beef tea cultures at 37° for periods varying from fifteen to thirty days, and shown to be toxic in both sterile and unsterilized conditions, provided that the sterilization was accomplished by means of heat, but when the beef tea culture was deprived of germs by filtration through porcelain, the germ-free filtrate could be injected into guinea-pigs in amounts of from eight to ten cubic centimeters without any other harm than that which would result from the introduction of an equal amount of sterile beef tea into the abdominal cavity of an animal. It follows from this that in the colon bacillus at least there is no soluble toxin formed. However, when the germ substance was obtained in large amount, free from constituents of the culture medium, extracted with alcohol and ether, dried and pulverized, and injected into animals in doses of from one to five milligrams, death resulted. This shows that the bacterial cell contains a toxin. In case of the colon bacillus the bacterial cell suspended in water and placed in a sealed tube may be heated in 180° C., for half an hour without loss of toxicity. Various attempts were made to extract the toxin from the cell by physical means. All kinds of solvents, including various salts of different strengths, were tried, without effect. These investigations led to the belief that the specific toxin of the colon bacillus is an integral part of the cell, and consists of a molecular group of the cell proteid. It was then found that when the dried colon cell substance was heated at the temperature of the water bath for a period not exceeding thirty minutes with one-per-cent. aqueous solution of sulphuric acid a certain constituent of the cell was split off by this process and passed into solution. The acid extract when filtered through porcelain or hard paper yields a voluminous pre-

precipitate when poured into three volumes of absolute alcohol. This precipitate, when collected, thoroughly washed until wholly free from acid, dried and pulverized, induces, when injected into animals, the symptoms and lesions of colon intoxication. So far this method of extracting the cellular toxins has been applied to the colon bacillus, and the bacilli of anthrax and diphtheria. These pathogenic organisms have all yielded, when treated with dilute sulphuric acid, toxins which, when injected into animals either subcutaneously or intraperitoneally, induce the symptoms and lesions which follow inoculation with virulent living cultures. The space limitation has already been exceeded, and we will have to omit a discussion of the properties of these intracellular toxins.

A BACTERIAL SOFT ROT OF CERTAIN CRUCIFEROUS PLANTS AND AMORPHOPHALLUS SIMLENSE.*

For several years the writers have had under observation a soft rot of certain cruciferous plants, particularly cabbage and cauliflower. During epidemics of black rot, *Pseudomonas campestris* (Pam.) Smith, in both cabbage and cauliflower it often happens that much damage is done by a soft rot. At first this soft rot was supposed to be merely a virulent form of black rot; but it was found that severe attacks of soft rot may occur in fields where there is little or no black rot. Especially is this true of seed cabbage. On Long Island the production of cabbage seed is an important industry, and one of the chief enemies to the crop is a soft rot which attacks the plants during winter storage in trenches and also at the time of blooming. In storage the plants are attacked just below the head. In the field this portion of the stem rots, causing the plant to suddenly wilt and die while in bloom.

In the cauliflower fields on Long Island one may find at any time during August, September and October plants which have suddenly collapsed with soft rot of the stem. From a plant thus affected the writers, in September,

* A preliminary report read before Section G of the A. A. A. S. at Pittsburgh, Pa., June 30, 1902.

1901, isolated an organism which in their notes was designated 0.2 E. Pure cultures of this organism were inoculated into cabbage and cauliflower plants in pots in the greenhouse in the following manner: A leaf springing from the fleshy portion of the stem was cut off close to the stem with a sterilized scalpel. Through the sterile surface thus formed the stem was punctured to the center by means of a needle which had been first sterilized and then dipped into a fresh culture of the organism. Finally the wound was smeared over with melted grafting wax. In this manner numerous plants of cabbage and cauliflower were inoculated at various times between March 10 and June 17. With one exception all of these plants became much rotten at the point of inoculation, whereas in the same number of check plants none showed any rot or discoloration whatever. The extent of the rotting seemed to depend largely upon the condition of the plant. On thrifty young plants it progressed with wonderful rapidity. Thrifty cabbage plants, two months old, nine inches high and with stems of the size of a lead pencil were so much rotted at the end of forty hours after inoculation that their own weight caused them to break over at the point of inoculation. On old, woody, slow-growing plants unmistakable signs of rot appear in from two to four days after inoculation; but in the majority of cases such plants are only checked in growth and not killed outright.

In most cases the rot first appears as a slight discoloration around the point of inoculation, works very rapidly for a few days, then stops. At first the rotten tissue is soft and mushy and watersoaked in appearance, but it soon dries and mostly disappears, leaving only a cavity lined with shreds of dry, blackened tissue.

Cabbage and cauliflower leaves inoculated in the petioles usually become broken over and soft rotten at the point of inoculation within forty-eight hours. Inoculations made in the blade of the leaf produce no marked results unless a large vein is punctured, in which case soft rot follows as with petiole inoculations. However, on the leaves of plants under bell-jars circular, dead, brown spots sometimes